U.S. Environmental Protection Agency
Science Advisory Board
Advisory Council on Clean Air Compliance Analysis,
Health Effects Subcommittee (HES)
Public Meeting
Marriott DC at Metro Center, 775 12th Street NW, Washington, DC, 20005
August 27-29, 2003
Agenda

Purpose: The purpose of the public meeting is to advise the Agency on its plan to develop a health effects assessment for the third in a series of statutorily mandated comprehensive analyses of the total costs and benefits of programs implemented pursuant to the Clean Air Act.

August 27, 2003

9:00-9:05	Opening of Subcommittee Meeting	Dr. Angela Nugent, Designated Federal Officer, SAB Staff
9:05-9:10	Welcome	Dr. Vanessa Vu, Director SAB Staff Office
9:10-9:25	Review of meeting purpose, agenda and HES Charge Questions (Attachment A to this Agenda); Introduction of Members	Dr. Bart Ostro, Chair
9:25-10:30	Brief Introduction to Analytical Plan, Status and Schedule for Study, Introduction to Project Team --- Presentation on the Relationship of the Analytical Plan to the Health Analyses in the Nonroad Diesel Draft RIA --- Agency Presentations and Discussion with Members on Issues #1-5 Identified by the Chair after the HES Public Teleconference on August 8. 2003 (Attachment B to this Agenda)	Mr. James DeMocker and Dr. Bryan Hubbell, US EPA Office of Air and Radiation
10:30-10:45	Break	

10:45-11:45	Continued Agency Presentations and Discussion	
11:45-12:00	Update on the August 25-26 Meeting of the Clean Air Scientific Advisory Committee's Revised Draft Air Quality Criteria Document PM Review Panel	Dr. Morton Lippmann
12:00-1:30	Lunch	
1:30-3:15	Charge Question 11: Ozone effects and covariation with Particulate Matter	Lead Discussants: Dr. Patrick Kinney, Dr. Morton Lippmann
	Concentration-Response function for Different Particulate Matter Sources	Lead Discussants: Dr. Morton Lippmann, Dr. Nino Kunzli,
	Extrapolation to Other Age Groups:	Lead Discussants: Dr. Morton Lippmann ,Dr. Rebecca Parkin
	Exposure Assessment (Use of Grids)	Lead Discussants: Dr. Patrick Kinney, Dr. Morton Lippmann
	Focus on Infant Effects	Lead Discussants: Dr. Nino Kunzli, Dr. Morton Lippmann
	Treatment of Asthma	Lead Discussants: Dr. Nino Kunzli, Dr. Bart Ostro
3:15-3:30	Break	
3:30-4:00	Charge Question 12 with Focus on Ozone	Lead Discussants: Dr. Patrick Kinney and Dr. Morton Lippmann
4:00-4:25	Charge Question 13: Baseline Data	Lead Discussants: Dr. Rebecca Parkin, Dr. Michael Kleinman
4:25-4:45	Charge Question 32: Plans for Evaluating Data Quality Inputs and Intermediate Data Products	Lead Discussants: Dr. Rebecca Parkin , Dr. Dale Hattis

4:45-5:05	Charge Question 33: Results aggregation regarding health effects	Lead Discussants: Dr. Michael Kleinman, Dr. Bart Ostro
5:05-5:25	Charge Question 34: Stratospheric Ozone Analysis	Lead Discussants: Dr. Morton Lippmann, Dr. Rebecca Parkin
5:24-5:45	Summary of Action Items; Preparation for Next Day	Dr. Bart Ostro
5:45	Adjourn	

August 28, 2003

8:30-8:35	Opening of Meeting/Administrative Business	Dr. Angela Nugent
8:35-9:00	Ethics Discussion	Ms.Peggy Love, Ethics Attorney, Office of General Counsel, EPA
9:00-9:10	Agenda Review	Dr. Bart Ostro
9:10-9:40	Agency Presentation on Issues #6 and 7 Identified by the Chair (Attachment B)	Mr. James DeMocker and Dr. Bryan Hubbell, US EPA Office of Air and Radiation
9:40-10:205	Charge Question 29: Expert Elicitation to Develop Probability-Based PM2.5 Concentration-Response Function for Premature Mortality	Lead Discussants: Mr. Fintan Hurley, Dr. Dale Hattis, Dr. John Evans,
10:20-11:00	Charge Question 30: Uncertainty and Ozone Mortality	Lead Discussants: Dr. Patrick Kinney, Dr. Morton Lippmann
11:00-11:15	Break	
11:15-11:35	Charge Question 11 with Focus on Effects of SO2, NO2, CO	Lead Discussants: Mr. Fintan Hurley, Dr. Michael Kleinman
11:35-11:55	Charge Question 12 with Focus on Morbidity Effects--Particulate Matter	Lead Discussants: Mr. Fintan Hurley (by teleconference), Dr. Bart Ostro
11:55-12:30	Charge Question 12 (a,b) and Charge Question 14: Alternative Methods for Estimating Particulate Matter-related Premature Mortality	Lead Discussants: Mr. Fintan Hurley (by teleconference), Dr. Nino Kunzli. Dr. Bart Ostro

12:30-1:45	Lunch	
1:45-2:30	Charge Question 15: Alternative Analysis for PM Control	Lead Discussants: (15a) Ms. Lauraine Chestnut (15b) Mr. Fintan Hurley (by teleconference), Dr. John Evans, Dr. Nino Kunzli (15c) Ms. Lauraine Chestnut
2:30-3:15	Charge Question 16: Latency and Cessation Lag: Time Delays in Benefits	Lead Discussants: Dr. Nino Kunzli; Dr. Morton Lippmann, Mr. Fintan Hurley (by teleconference)
3:15-3:30	Break	
3:30-4:45	Charge Question 17: Questions related to presentation of alternative estimate of benefits as well as the base estimate	Lead Discussants: (17a) Dr. Bart Ostro (17b,c) Ms. Lauraine Chestnut (17d-i) Dr. Nino Kunzli (17d-ii) Dr. Michael Kinney (17d-iii) Ms. Lauraine Chestnut
4:45-5:30	Charge Questions 35 and 36: Air Toxics	Lead Discussants: Dr. Dale Hattis, Dr. Michael Kleinman
5:30-5:45	Summary of Action Items/Preparation for Next Day	Dr. Bart Ostro
5:45	Adjourn	

August 29, 2003

9:00-9:05	Opening of Meeting/Administrative Business	Dr. Angela Nugent
9:05-9:15	Agenda Review	Dr. Bart Ostro
9:15-10:15	Discussion of Major Themes Across all Health Assessment Topics	All Subcommittee Members
10:15-12:00	Time for Drafting Report	
12:00-1:00	Lunch	
1:00-2:15	Discussion of Report Issues	All Subcommittee Members
2:15-2:30	Summary of Action Items/Preparation for September Council Meeting	Dr. Bart Ostro
2:30	Adjourn	

Attachment A
Charge Questions for the Health Effects Subcommittee
Excerpted from the List of 37 Charge Questions (Revised as of July 3, 2003)
Provided to the Advisory Council on Clean Air Compliance Analysis

Chapter 6: Human Health Effects Estimation

11. Does the Council support the plans described in chapter 6 for estimating, evaluating, and reporting changes in health effect outcomes between scenarios? If there are particular elements of these plans which the Council does not support, are there alternative data or methods the Council recommends?

12. EPA seeks advice from the Council regarding the technical and scientific merits of incorporating several new or revised endpoint treatments in the current analysis. These health effect endpoints include:
    a. Premature mortality from particulate matter in adults 30 and over, PM (Krewski et al., 2000);
    b. A PM premature mortality supplemental calculation for adults 30 and over using the Pope 2002 ACS follow-up study with regional controls;
    c. Hospital admissions for all cardiovascular causes in adults 20-64, PM (Moolgavkar et al., 2000);
    d. ER visits for asthma in children 0-18, PM (Norris et al., 1999);
    e. Non-fatal heart attacks, adults over 30, PM (Peters et al., 2001);
    f. School loss days, Ozone (Gilliland et al., 2001; Chen et al., 2000);
    g. Hospital admissions for all respiratory causes in children under 2, Ozone (Burnett et al., 2001); and,
    h. Revised sources for concentration-response functions for hospital admission for pneumonia, COPD, and total cardiovascular: Samet et al., 2000 (a PM10 study), to Lippmann et al., 2000 and Moolgavkar, 2000 (PM2.5 studies).

13. EPA seeks advice from the Council regarding the merits of applying updated data for baseline health effect incidences, prevalence rates, and other population characteristics as described in chapter 6. These updated incidence/prevalence data include:
    a. Updated county-level mortality rates (all-cause, non-accidental, cardiopulmonary, lung cancer, COPD) from 1994-1996 to 1996-1998 using the CDC Wonder Database;
    b. Updated hospitalization rates from 1994 to 1999 and switched from national rates to regional rates using 1999 National Hospital Discharge Survey results;
    c. Developed regional emergency room visit rates using results of the 2000 National Hospital Ambulatory Medical Care Survey;
    d. Updated prevalence of asthma and chronic bronchitis to 1999 using results of the National Health Interview Survey (HIS), as reported by the American Lung Association (ALA), 2002;
    e. Developed non-fatal heart attack incidence rates based on National Hospital Discharge Survey results;

f. Updated the national acute bronchitis incidence rate using HIS data as reported in ALA, 2002, Table 11;
g. Updated the work loss days rate using the 1996 HIS data, as reported in Adams, et al. 1999, Table 41;
h. Developed school absence rates using data from the National Center for Education Statistics and the 1996 HIS, as reported in Adams, et al., 1999, Table 46.
1. Developed baseline incidence rates for respiratory symptoms in asthmatics, based on epidemiological studies (Ostro et al. 2001; Vedal et al. 1998; Yu et al; 2000; McConnell et al., 1999; Pope et al., 1991).

14. EPA plans to initiate an expert elicitation process to develop a probability-based method for estimating changes in incidence of PM-related premature mortality. Plans for this expert elicitation are described in chapter 9 of this blueprint, and a separate charge question below requests advice from the Council pertaining to the merits of the design of this expert elicitation. EPA recognizes, however, the possibility that this expert elicitation process may not be fully successful and/or may not be completed in time to support the current 812 analysis. Therefore, in order to facilitate effective planning and execution of the early analytical steps which provide inputs to the concentration-response calculations, EPA seeks advice from the Council regarding the scientific merits of alternative methods for estimating the incidences of PM-related premature mortality, including advice pertaining to the most scientifically defensible choices for the following specific factors:
    a. Use of cohort mortality studies, daily mortality studies, or some combination of the two types of studies
    b. Selection of specific studies for estimating long-term and/or short-term mortality effects
    c. Methods for addressing –either quantitatively or qualitatively– uncertain factors associated with the relevant concentration-response function(s), including
    i. Shape of the PM mortality C-R function (e.g., existence of a threshold),
    ii. PM causality,
    iii. PM component relative toxicity, and
    iv. PM mortality effect cessation lag structure
    v. Cause of death and underlying health conditions for individuals dying prematurely due to chronic and/or short term exposures to particulate matter
    vi. The use of ambient measures of exposure for estimating chronic health effects, given recent research reviewed in the NAS (2002) report that questions the implications of using ambient measures in cohort studies

15. EPA estimates of benefit from particulate control may underestimate the impact of nonfatal cardiopulmonary events on premature mortality and life expectancy. For the base analyses, which rely on cohort evidence, the limited follow-up periods for the cohorts may not fully capture the impacts of nonfatal cardiovascular events on premature mortality later in life. For the alternative analyses –including cost-effectiveness analyses– which rely more on acute studies

and life-expectancy loss, the years of life are estimated only for fatal events. Yet nonfatal events such as myocardial infarction reduce a person's life expectancy by a substantial percentage.

a. Do you agree that EPA, in the 812 analyses, should adjust benefit estimates to account for the mortality effects of non-fatal cardiovascular and respiratory events?
b. What medical studies and mathematical models of disease might be useful to review or use if EPA moves in this direction?
c. When the nonfatal events are valued in economic terms, should EPA assume that the published unit values for morbidity already account for the life-expectancy loss or should an explicit effort be made to monetize the resulting longevity losses?

16. In recent EPA rulemakings, EPA's "base estimate" of benefit from PM control has been based on cohort epidemiological studies that characterize the chronic effects of pollution exposure on premature death as well as capturing a fraction of acute premature mortality effects. If these chronic effects occur only after repeated, long-term exposures, there could be a substantial latency period and associated cessation lag. As such, a proper benefits analysis must consider any time delay between reductions in exposure and reductions in mortality rates. For the acute effects, such as those considered in EPA's alternative benefit analyses, the delays between elevated exposure and death are short (less than two months), and thus time-preference adjustments are not necessary.

a. In the previous 812 analysis and in recent rulemakings, EPA assumed a weighted 5-year time course of benefits in which 25% of the PM-related mortality benefits were assumed to occur in the first and second year, and 16.7% were assumed to occur in each of the remaining 3 years. Although this procedure was endorsed by SAB, the recent NAS report (2002) found "little justification" for a 5-year time course and recommended that a range of assumptions be made with associated probabilities for their plausibility. Do you agree with the NAS report that EPA should no longer use the deterministic, 5-year time course?
b. One alternative EPA is considering is to use a range of lag structures from 0 to 20-30 years, with the latter mentioned by NAS in reference to the Nyberg et al PM lung cancer study, with 10 or 15 years selected as the mid-point value until more definitive information becomes available. If this simple approach is used, should it be applied to the entire mortality association characterized in the cohort studies, or only to the difference between the larger mortality effect characterized in the cohort studies and the somewhat smaller effect found in the time series studies of acute exposure? Should judgmental probabilities be applied to different lags, as suggested by NAS?
c. Another option under consideration is to construct a 3-parameter Weibull probability distribution for the population mean duration of the PM mortality cessation lag. The Weibull distribution is commonly used to represent probabilities based on expert judgment, with the 3-parameter version allowing the shaping of the probability density function to match expected low, most likely, and expected high values. EPA is still

considering appropriate values for the low, most likely, and expected high values –and therefore for the Weibull shape and location parameters– and EPA is interested in any advice the Council wishes to provide pertaining to the merits of this approach and/or reasonable values for the probability distribution.

17. In support of Clear Skies and several recent rule makings the Agency has presented an Alternative Estimate of benefits as well as the Base Estimate. EPA developed the Alternative Estimate as an interim approach until the Agency completes a formal probabilistic analysis of benefits. NAS (2002) reinforced the need for a probabilistic analysis. The Alternative Estimate is not intended as a substitute method and needs to be considered in conjunction with the Base Estimate. Presentation of Base and Alternative estimates in the 812 Report may not be necessary if the probability analysis planned for the 812 Report is successful. While the Base Estimate assumes that acute and chronic mortality effects are causally related to pollution exposure, the Alternative Estimate assumes only acute effects occur or that any chronic effects are smaller in size than assumed in the Base Estimate. The Council's advice is sought on the following matters:

    a. It has been noted by some particle scientists that the size of estimates based on time series studies that incorporate a distributed lag model, accounting for effects of 30 to 60 days after elevated exposure, may be similar in size to some interpretations of the results from the cohort studies. Does the Council agree that it is a reasonable alternative to use an estimate of the concentration-response function consistent with this view? If the Council agrees with the assumption, can it suggest an improved approach for use in an Alternative Estimate? The agency also seeks advice on appropriate bounds for a sensitivity analysis of the mortality estimate to be used in support of the Alternative Estimate.

    b. An assumption that a specific proportion of the PM-related premature mortality incidences are incurred by people with pre-existing Chronic Obstructive Pulmonary Disease (COPD) and that these incidences are associated with a loss of six months of life, regardless of age at death. If these values are not valid, what values would be more appropriate? Do you recommend a sensitivity analysis of 1 to 14 years (with the latter based on standard life tables), as included in the draft regulatory impact analysis of the proposed Nonroad diesel rule?

    c. An assumption that the non-COPD incidences of PM-related premature mortality are associated with a loss of five years of life, regardless of age at death. If these values are not valid, what values would be more appropriate? Do you recommend a sensitivity analysis of 1 to 14 years (with the latter based on standard life tables), as included in the draft regulatory impact analysis of the proposed Nonroad diesel rule?

    d. Additional quantified and/or monetized effects are those presented as sensitivity analyses to the primary estimates or in addition to the primary estimates, but not included in the primary estimate of total monetized benefits. While no causal mechanism has been identified for chronic asthma and ozone exposure, there is suggestive epidemiological evidence.

i. Two studies suggest a statistical association between ozone and new onset asthma for two specific groups: children who spend a lot of time exercising outdoors and non-smoking men. We seek SAB comment on our approach to quantifying new onset asthma in the sensitivity analyses.
ii. Premature mortality associated with ozone is not currently separately included in the primary analysis because the epidemiological evidence is not consistent. We seek SAB comment on our approach to quantifying ozone mortality in the sensitivity analyses.
iii. Does the Council agree that there is enough data to support a separate set of health impacts assessment for asthmatics? If so, does the approach proposed by the Agency address the uncertainty in the literature?

Chapter 9: Uncertainty Analysis

29. Does the Council support the plans described in chapter 9 for the expert elicitation pilot project to develop a probability-based PM2.5 C-R function for premature mortality, including in particular the elicitation process design? If the Council does not support the expert elicitation pilot project, or any particular aspect of its design, are there alternative approaches the Council recommends for estimating PM-related mortality benefits for this analysis, including in particular a probabilistic distribution for the C-R function to reflect uncertainty in the overall C-R function and/or its components?

30. EPA plans to develop estimates of an independent mortality effect associated with ozone, as described in chapter 9. Does the Council support the use of the most recent literature on the relationship between short-term ozone exposure and daily death rates, specifically that portion of the literature describing models which control for potential confounding by PM2.5? Does the Council agree with the use of that literature as the basis for deriving quantified estimates of an independent mortality impact associated with ozone, especially in scenarios where short-term PM2.5 mortality estimates are used as the basis for quantifying PM mortality related benefits? Does the Council support the plans described in chapter 9 for the pilot project to use this literature to develop estimates of the ozone related premature mortality C-R function using the three alternative meta-analytic approaches? If the Council does not support this pilot project, or any particular aspect of its design, are there alternative approaches to quantifying ozone-related premature mortality which the Council recommends?

Chapter 10: Data Quality and Intermediate Data Products

32. Does the Council support the plans described in chapter 10 for evaluating the quality of data inputs and analytical outputs associated with this study, including the planned publication of intermediate data products and comparison of intermediate and final results with other data or estimates? If the Council does not support these plans, are there alternative approaches, intermediate data products,

data or model comparisons, or other data quality criteria the Council reommends? Please consider EPA's Information Quality Guidelines in this regard.

Chapter 11: Results Aggregation and Reporting

33. Does the Council support the plans described in Chapter 11 for the aggregation and presentation of analytical results from this study? If the Council does not support these plans, are there alternative approaches, aggregation methods, results presentation techniques, or other tools the Council recommends?

Appendix D: Stratospheric Ozone Analysis

34. Does the Council support the plans describe in Appendix D for updating the estimated costs and benefits of Title VI programs? If the Council does not support these plans, are there alternative data, models, or methods the Council recommends?

Appendix E: Air Toxics Case Study

35. Does the Council support the plans described in Appendix E for the benzene case study, including the planned specific data, models, and methods, and the ways in which these elements have been integrated? If the Council does not support these plans, are there alternative data, models, or methods the Council recommends?

36. A cessation lag for benzene-induced leukemia is difficult to estimate and model precisely due to data limitations, and EPA plans to incorporate a five-year cessation lag as an approximation based on available data on the latency period of leukemia and on the exposure lags used in risk models for the Pliofilm cohort (Crump, 1994 and Silver et al., 2002). Does the SAB support adoption of this assumed cessation lag? If the Council does not support the assumed five-year cessation lag, are there alternative lag structures or approaches the Council recommends?

Appendix H: Meta-analysis of VSL

37. Does the Council support including the Kochi et al. (2002) meta-analysis as part of a the larger data base of studies to derive an estimate for the value of avoided remature mortality attributable to air pollution? Are there additional data, models, or studies the Council recommends? Does the SAB think that EPA should include Kochi et al. 2003 if not accepted for publication in a peer reviewed journal by the time the final 812 report is completed?

Attachment B
Issues identified by the Chair after the HES Public Teleconference on August 8, 2003:

1. EPA should provide a brief review of their approach to uncertainty. Many of the aspects of the alternative analyses could be subsumed within a probabilistic framework and combined with the base analysis. Will the Agency be using this approach prior to the completion of the pilot subjective probability studies described in the Uncertainty section? For example, ozone-related mortality could be part of the base analysis using a probabilistic weight.

2. Is the Agency convinced that estimated effects (both "old" and new endpoints) of ozone, NO2, SO2 are independent of PM effects?

3. The focus seems to be on PM2.5 rather than PM10 in several of the endpoints. Are all PM10 benefits included within PM2.5?

4. What is the Agency's proposed approach for assessing of ozone-related mortality?

5. Are there mathematical models of disease that EPA is considering to address the issue the issue of mortality effects of non-fatal CV events? Will EPA adjust benefit analysis to account for these effects?

6. How is the Agency planning to use and combine the cohort and time-series studies to estimate mortality effects? Which studies will be used and what range of effect estimates?

7. What is the Agency's proposed approach for latency and lag estimates in relating reductions in long-term exposure to mortality rates and for life years lost from COPD and non-COPD related deaths? What information is being used to develop these estimates?